Rapid speciation and chromosomal evolution in mammals

(evolutionary rates/effective population size/vertebrates/cytogenetic isolating mechanisms/social structuring)

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ABSTRACT   To test the hypothesis that population subdivision into small demes promotes both rapid speciation and evolutionary changes in gene arrangement by inbreeding and drift, we estimated rates of speciation and rates of chromosomal evolution in 225 genera of vertebrates. Rates of speciation were estimated by considering the number of living species in each genus and the fossil record of each genus as well as information about extinction rates. Speciation rate was strongly correlated with rate of chromosomal evolution and average rates of speciation in lower vertebrate genera were one-fifth those in mammalian genera. Genera with high karyotypic diversity and rapid speciation rates may generally have small effective population size (N_e), whereas large N_e values may be associated with karyotypically uniform genera and slow rates of speciation. Speciation and chromosomal evolution seem fastest in those genera with species organized into clans or harems (e.g., some primates and horses) or with limited adult vagility and juvenile dispersal, patchy distribution, and strong individual territoriality (e.g., some rodents). This is consistent with the above hypothesis regarding the evolutionary importance of demes.

Among evolutionary biologists there is a widespread acceptance of the hypothesis that small populations are essential for rapid speciation (1–7). According to this hypothesis, new species may arise from populations initially founded by a small number of individuals isolated on the periphery of the range of the parental species—the founder principle (3–5)—or in small demes maintained by social structuring and ecology (7, 8). Widespread also is the idea that small populations are essential for chromosomal evolution, and some authors suggest that the two processes—speciation and chromosomal evolution—may be causally related (7–12). It is now possible to test these ideas quantitatively because methods have been developed recently for estimating both rates of speciation (13, 14) and rates of evolutionary change in gene arrangement (8, 15). Unfortunately, few estimates of effective population size are available. Hence, it is not easy to make a direct examination of the relationship between population size and rates of evolution. It is possible, however, to examine quantitatively the relationship between rate of speciation and rate of chromosomal evolution. If both processes are dependent on the occurrence of small demes, the two rates should be correlated. Such a correlation was found in higher plants (14).

The present article deals with vertebrates. Rates of chromosomal evolution vary greatly in these animals, being an order of magnitude higher in most mammals than in most other vertebrates (8, 15). It was important, therefore, to estimate rates of speciation in a wide variety of vertebrates. The results of such estimates are reported below and compared with the results of an improved method for estimating rate of chromosomal evolution.

METHODS

Rates of Chromosome Evolution. In an earlier analysis of chromosome evolution (8), a conservative approach was used to estimate rates of karyotypic change. This method could seriously underestimate the number of karyotypic changes, however, because it dealt only with the number of karyotypes observed. It did not estimate the minimum number of chromosomal mutations which must have occurred to produce the observed range of karyotypes.

We have now recalculated the rates of change in karyotype with a revised equation:

$$r' = \sum_{i=1}^{G} (a_i + b_i) \Big/ \sum_{i=1}^{G} t_i \qquad [1]$$

in which G is the number of genera examined karyotypically within a major taxonomic group and t is the time of first appearance of a genus in the fossil record [in millions of years (Myr) ago]. The symbol a is defined as $(c - d)/n$, in which c is the highest number of chromosomes and d is the lowest number of chromosomes per haploid genome within a genus; n is the number of species examined karyotypically within that genus. Likewise, b is defined as $(e - f)/n$, in which e is the highest number of chromosome arms (fundamental number) and f is the smallest number of chromosome arms per haploid genome within a genus.

The revised method is based on the assumption that one mutation can change either the haploid chromosome number by ±1 or the haploid arm number by ±1. Because some cases of polyploid species are known in fishes, amphibians, and reptiles, this method could over estimate the total rates of karyotypic evolution; a single event could change the ploidy level. For this reason, a change in ploidy level was counted as only one event.

This new method is still somewhat inadequate because it detects only a fraction of all chromosomal rearrangements fixed. Fusions, fissions, and whole-arm inversions are detected, whereas all paracentric inversions, most reciprocal translocations, and many pericentric inversions are not. All of the latter are unquestionably important in chromosome evolution and can serve as sterility barriers in speciation. The method also does not compensate for the differential occurrence of specific rearrangement types in given lineages, termed "karyotypic orthoselection" by White (11). However, groups that are extremely conservative in chromosome evolution are also conservative with respect to changes in banding pattern (16). By contrast, rapidly evolving mammalian groups (e.g., primates) have undergone extensive changes in banding pattern (17). The fraction of chromosomal rearrangements detected by our method may therefore be roughly proportional to the total. In addition, we are aware that our methods obscure intragroup variability in rates.

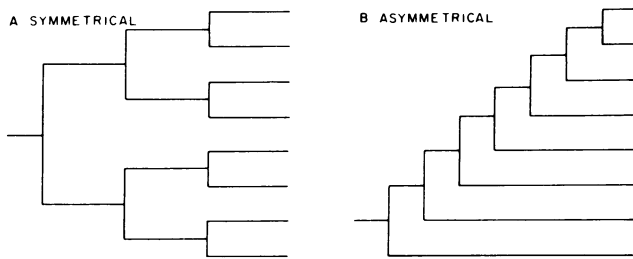Abbreviation: Myr, millions of years.

FIG. 1.   Two branching patterns that produce the same number of species (8) but require a different number of speciation events (h) per lineage. In the symmetrical model (A) there are 3 speciation events in each lineage, whereas there is an average of 4.4 speciation events per lineage in the asymmetrical model (B).

As in a previous analysis (8), we restricted attention to those polytypic genera for which chromosome number, arm number, and age of first appearance in the fossil record are known. In total, 1511 species, representing 225 genera, were examined. We recognize that the sampling of lower vertebrate genera is less adequate than that in mammals. Most data were obtained from Denton (18), Chiarelli and Capanna (19), and Patton and Mascarello.[§] Birds were not included in this analysis because of the problem of counting accurately the numbers of chromosomes and arms.

The age of each genus is based on the time of first appearance in the geological record of taxa assigned to that genus. These determinations were made by examining numerous reports in the paleontological literature and by consulting paleontologists in California and Texas. Absolute ages were assigned to the geological record as per Savage (20). For some groups the time of the first appearance is probably an accurate estimate of the absolute generic age. For others, particularly many bats, rodents, and other microfaunal groups, this is a severe underestimate. No attempt has been made to correct the data for these inadequacies.

Rates of Speciation. Following Stanley (13), the rate at which new species arise, S, was calculated by $S = R + E$, where, within a given genus, R is the net rate at which new species have arisen and E is the average rate at which species have gone extinct over a defined period of time in the past.

Calculations of R depend on the pattern of branching of lineages within a given genus. For most genera, however, the actual pattern of branching is not established. We therefore considered the two contrasting models of branching depicted in Fig. 1. In the symmetrical model (A) the number of speciation events is the same along every lineage, in this case 3, and can be described by

$$g = 2^h \text{ or } h = 3.3 \log g \qquad [2]$$

in which g is the number of extant species per genus and h is the number of speciation events per lineage. In the asymmetrical model (B), the number of speciation events per lineage is not constant but varies from 1 to $g - 1$. The average number of speciation events per lineage for such a phylogenetic tree is given by

$$h = \frac{g + 1}{2} - \frac{1}{g} \qquad [3]$$

This estimate of h exceeds that based on Eq. 2.

To decide which model is the more realistic, we estimated h directly from several known phylogenetic trees. The results suggest that Eq. 2 is more realistic than Eq. 3 for groups such as frogs and pinnipeds, which have low rates of chromosomal

evolution. Hence, for the purposes of further computations, we used Eq. 2 to estimate the net speciation rates in all genera examined. When viewing the results of our calculations (see below), it is worth recalling that our method probably underestimates the rate of speciation in most of those groups whose chromosomal evolution has been very fast.

The mean value of the net speciation rate, R, was calculated for each major group of vertebrates by

$$R = \sum_{i=1}^{G} h_i \Big/ \sum_{i=1}^{G} t_i \qquad [4]$$

in which G is the number of genera examined karyotypically within the group; thus the h values calculated for each genus are summed and divided by the summed time range (t) for those genera. Data on the number of species per genus were obtained from Gorham (21), Duellman,[¶] and Anderson and Jones (22).

Because extinction rates (E) are difficult to estimate reliably for species within a given genus, we estimated average rates of extinction for species within major groups of vertebrates. Consider a major group, such as frogs. We ask how far back in time must one go to find strata in which 50% of the frog fossils belong to extant species. This time may be designated $D/2$, where D is a minimum estimate of the mean duration time for species in that major group (13). The mean extinction rate (E) for species within the group is then taken to be $1/D$. Extinction rates for most major groups of mammals were calculated from fossil data summarized by Kurtén (23). The values for whales, marsupials, and lower vertebrates are based on an extensive survey of diverse papers in the paleontological literature.

By adding the E value to the R value for a major group of vertebrates, one gets an estimate of the true speciation rate (S), which we refer to as the corrected speciation rate. The corrected speciation rate is thus based on the extant species diversity in each genus as well as on a rough estimate of the probable rate at which species have become extinct since the origin of the genus.

Because of space limitations, we cannot here present all the data on which the evolutionary rate calculations are based. A list of all the genera used is available upon request, however. The list includes, for each genus, the values used for $c - d, e - f, n, g, h$, and t.

RESULTS

The new estimates of rates of chromosomal evolution within living genera appear in Table 1 for 16 major groups of vertebrates. These estimates are consistent with those published before (8) in two main respects. First, compared to the average mammalian genus, lower vertebrate genera seem to have evolved very slowly in karyotype. Second, there is diversity among mammals—whales, for example, having evolved more slowly than primates.

The absolute values given here are similar to those reported earlier (8) for lower vertebrates and whales. However, the new estimates for the most rapidly evolving groups of mammals are several times higher than those reported before (8). So, the present analysis serves to emphasize the conclusion that many mammals—especially primates and horses—have evolved extraordinarily rapidly at the chromosomal level.

Our estimates of speciation rates within living genera appear in Table 2. The lowest net rates of speciation (R) occur in lower vertebrates and the highest rates in mammals. Extinction rates (E) have also been higher, on the average, in mammals than in

§ Patton, J. L. & Mascarello, J. T. (1977) in *Animal Cytogenetics, Vol. 4, Chordata 4: Mammalia II: Placentalia II, I: Rodentia,* ed. John, B. (Gebruder Bortrager, Stuttgart), in preparation.

¶ Duellman, W. E. (1961) "A classification of the recent Reptilia," unpublished manuscript.

Table 1. Rates of chromosome evolution in extant genera of vertebrates

Group	Number of genera examined (G)	Average age of genera, Myr	Karyotypic changes/ lineage per Myr Chromosome number	Karyotypic changes/ lineage per Myr Arm number
Mammals				
Horses	1	3.5	0.609	0.786
Primates	13	3.8	0.333	0.413
Lagomorphs	3	5.0	0.230	0.403
Rodents	50	6.0	0.178	0.253
Artiodactyls	15	4.2	0.364	0.197
Insectivores	7	8.1	0.074	0.113
Marsupials*	15	5.6	0.052	0.124
Carnivores	10	12.9	0.042	0.036
Bats	15	9.0	0.028	0.031
Whales	2	6.5	0.000	0.025
Average		6.5	0.129	0.166
Other vertebrates				
Lizards	16	20.1	0.027	0.031
Snakes	14	12.1	0.007	0.041
Teleosts	12	5.7	0.003	0.026
Frogs	15	26.4	0.011	0.012
Salamanders	11	23.4	0.006	0.008
Turtles and crocodiles	14	45.2	0.0006	0.0016
Average		22.1	0.009	0.020

*Previously published estimates (8) failed to include data from Macropodidae (kangaroos, wallabies, etc.).

Table 2. Rates of speciation in extant genera of vertebrates

Group	Net speciation rate (R)	Mean species duration time (D)	Extinction rate (E)	Corrected speciation rate (S)
Mammals				
Horses	0.80	0.5	2.0	2.8
Primates	0.60	0.5	2.0	2.6
Lagomorphs	0.81	1.2	0.8	1.6
Rodents	0.56	1.0	1.0	1.6
Artiodactyls	0.48	1.0	1.0	1.5
Insectivores	0.42	1.0	1.0	1.4
Marsupials	0.40	1.6	0.6	1.0
Carnivores	0.25	1.2	0.8	1.1
Bats	0.44	3.2	0.3	0.7
Whales	0.19	4.0	0.25	0.4
Average	0.50	1.5	1.0	1.5
Other vertebrates				
Lizards	0.23	26.0	0.04	0.3
Snakes	0.33	16.0	0.06	0.4
Teleosts*	0.41	—	—	—
Frogs	0.18	16.0	0.06	0.2
Salamanders	0.12	5.0	0.2	0.3
Turtles and crocodiles	0.06	5.0	0.2	0.3
Average	0.24	14.0	0.1	0.3

*Insufficient fossil information for calculation of extinction rates.

karyotypic rate (0.4–1.4 events/lineage per Myr) approach the rate of speciation (1.6–2.8 new species/lineage per Myr). By contrast, for the slowly evolving groups, the mean karyotypic rate (0.03 events/lineage per Myr) is 1/10 the mean speciation rate (0.3 new species/lineage per Myr). Unfortunately, because extinction rates within each genus examined are not known, we cannot analyze this relationship on a genus-by-genus basis.

other vertebrates. In consequence, the corrected rates of speciation (S) are substantially higher for most mammalian groups than for lower vertebrates. Primates and horses seem to have experienced especially high corrected rates of speciation.

We have plotted the average rate of speciation (S) against the average rate of karyotypic evolution (r') for each major group of vertebrates, as shown in Fig. 2. A highly significant correlation between the two rates exists ($r^2 = 0.831$). Yet the relationship between these two quantities is not simple. For the most rapidly evolving groups, the absolute values of the
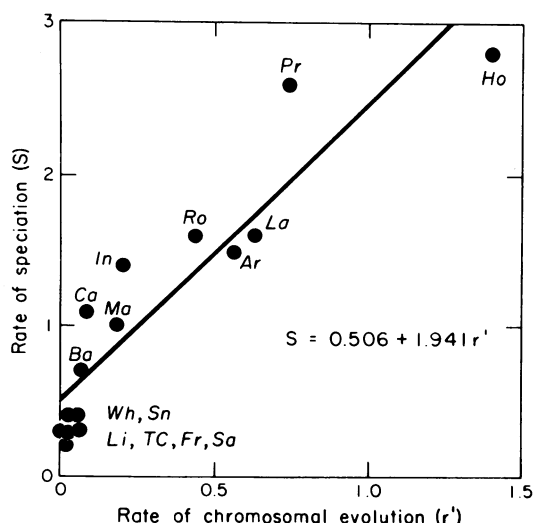

FIG. 2. Relationship between corrected speciation rate (S) and rate of karyotypic change (r') for major groups of vertebrates. Abbreviations: Ar, artiodactyls; Ba, bats; Ca, carnivores; Fr, frogs; Ho, horses; In, insectivores; La, lagomorphs; Li, lizards; Ma, marsupials; Pr, primates; Ro, rodents; Sa, salamanders; Sn, snakes; TC, turtles and crocodilians; Wh, whales.

DISCUSSION

The association between high rates of speciation and of chromosomal evolution is consistent with the hypothesis, outlined in the *Introduction,* that both processes are accelerated by the subdivision of a species into demes. While such a correlation does not necessarily imply a cause and effect relationship between the incidence of demes and the two processes, this possibility deserves serious consideration.

The observation that speciation has occurred at an appreciable rate (one new species per lineage per 3 Myr) in the virtual absence of chromosomal evolution (see Fig. 2) should also be examined in light of the above possible relationship. According to the classical allopatric theory, speciation occurs slowly when a species becomes divided by a geographic barrier into two large populations. In such large populations, karyotypic mutations, which often have reduced fitness in the heterozygous state (24), have essentially no chance of surviving and becoming fixed. This type of speciation may have predominated within most of the extant genera examined in the groups shown in the lower left part of Fig. 2.

It is important, therefore, to obtain direct evidence as to whether small demes are characteristic of those genera whose speciation rates and rates of chromosomal evolution are especially high.

Effective Population Size. The factors that produce population subdivision and lower the effective population size (N_e) include low vagility (25), strong territoriality, patchiness of the

environment (26), and social structuring. Small N_e values are especially likely in temporally stable social units that contain a single reproductive male and several breeding females, as in harems or clans (7, 8). Social organizations of this sort are found in many mammals and may be the only means by which large, mobile mammals can maintain the population subdivision required for the observed rapid rates of speciation and karyotypic evolution without the necessity for strict geographic isolation (7, 8).

Unfortunately, few precise estimates are available for N_e in mammals or other vertebrates, so that hypotheses regarding N_e can only be tested by less satisfactory, circumstantial means for the moment. We can ask, however, whether those genera that display wide karyotypic diversity are also those in which subdivision of populations into small demes is most marked.

In primates, which as a group have about the highest speciation rate among mammals (Table 2), the karyotypically diverse vervet monkeys and their relatives (genus *Cercopithecus*) seem to contrast sharply in patterns of sociality with the chromosomally uniform baboons (*Papio*). *Cercopithecus* species generally exist in small troops consisting of a single adult male and several adult females, whereas species of *Papio* are organized into multi-male troops, usually of quite large numbers (27). The difference in N_e between the taxa is likely to be about an order of magnitude, and we suggest that this difference may be largely responsible for the apparent zero rate of chromosome change in baboons. As a result, species of *Cercopithecus* are often sympatric, but if so, are invariably chromosomally distinct with interbreeding in nature very rare. On the other hand, all baboon species are normally allopatric and, where contact has recently been established, hybridization is common, suggesting a lack of complete reproductive isolation between species (28).

Complex levels of sociality are not always a prerequisite, however, for small population size in mammalian species. In many rodents, for example, a combination of asociality manifested in strong individual territoriality, limited adult vagility and juvenile dispersal distance, and patchy distributions can produce the small deme size necessary for rapid chromosomal fixation (29). While specific data on the types of social structuring within various rodent genera are generally not available, the strongly fossorial groups [e.g., pocket gophers (*Thomomys*, *Geomys*, and their relatives), mole rats (*Spalax*), and tucotucos (*Ctenomys*)] are considered to represent the epitome of this pattern (30, 31). These genera are also among the most chromosomally diverse rodents known (32–34).

A combination of social structuring with low vagility and environmental patchiness may be responsible for rapid chromosome evolution and speciation in mice. As pointed out elsewhere (7, 8), many rodents also have sociality patterns similar to the harem formation of horses (35, 36) and *Cercopithecus* described above. For example, populations of house mice (*Mus musculus*) are subdivided into small family groups of four to seven reproductively active individuals with one dominant male (29). Group territories are defended and, while young individuals disperse from these social groups, there is little interchange between them (37, 38). Even in the confines of a single barn, genetically distinct demes of house mice have been recognized (39). Similar breeding structures have been reported for *Apodemus* (40) and *Peromyscus* (41). In the latter, N_e has been estimated to range from 12 to 99, assuming various densities of mature mating individuals. Chromosomal variation is in general the rule both within and between species of these genera. Indeed, the multiplicity of chromosomal rearrangements recently found in house mouse populations in alpine valleys of Switzerland and Italy (42, 43) and the incredible array

of karyotypes encountered among African species belonging to the subgenus *Leggada* of *Mus* (44) are most likely the product of social organization interacting with environmental patchiness in the production of sufficiently small N_e for these changes to occur.

Groups with intermediate evolutionary rates (i.e., marsupials, carnivores) may have speciated predominantly by the classical allopatric mechanism, and to a lesser extent by stasipatric speciation. Species in these groups are mostly solitary in behavior and seldom establish small demes. For example, many carnivores lead solitary lives and when social units are formed they are frequently structured in such a way that inbreeding and drift are unlikely to occur. Lion prides, for instance, are organized around a group of reproductive females. Although only one or two males are usually associated with a pride at any one time, there is a constant turnover in males, with few remaining for more than two years in any pride (45). All other cat species spend their lives alone except at the time of mating or while rearing young. The N_e of cats is therefore large even in lions and this fact is reflected in the apparent low rate of chromosome evolution in this group (46). This example demonstrates that not all forms of social structuring in mammals and other vertebrates will necessarily lead to rapid chromosome evolution and speciation. Only when the social structuring and behavior results in small N_e will evolutionary rates be accelerated.

Whales and bats are apparently exceptional among mammals. During the past 10 million years, rates of speciation, karyotypic evolution and morphological evolution within extant genera of whales and bats have been low. This is due probably to their great dispersal power, which interferes with maintenance of small demes. Also in whales there is a tendency to aggregate into large pods organized around nursing females (47).

Our hypothesis predicts that, among those many lower vertebrates whose tempo of karyotypic evolution and speciation has generally been low, N_e values will be found to exceed those for most mammals. Unfortunately, there is little satisfactory information on N_e in lower vertebrates (47).

To obtain satisfactory estimates of N_e will be a major undertaking for population biologists. This will require many more genetically oriented field studies of natural populations undisturbed by humans and representing groups that have contrasting rates of chromosomal evolution. Most published studies of heterozygosity levels at loci coding for proteins may not have met this requirement. Nevertheless, it is notable that these levels seem especially low in mammals (48, 49). This is consistent with our hypothesis.

Karyotypic Facilitation of Speciation and Adaptive Evolution. Although the correlation between karyotypic evolution and speciation, shown in Fig. 2, might be ascribed simply to the possibility that both processes are accelerated by population subdivision, it seems reasonable to think that there may be additional ways in which the two processes are related. In particular, Grant (50) and White (10, 11) pointed out three possible ways in which fixation of karyotypic mutations could facilitate speciation and adaptive evolution at the organismal level.

(*i*) A karyotypic mutation that has become fixed in a given deme can act as a sterility barrier, impeding gene flow between that deme and others. Thus, the mutant karyotype functions at the population level as a cytogenetic reproductive isolating mechanism. Speciating by this process has been termed "stasipatric speciation" by White (10).

(*ii*) The mutation acts at a molecular level as a regulatory mutation, producing an altered pattern of gene expression that

results in an organism with a new and fitter phenotype. For an example of how gene rearrangement can alter gene expression see Zieg *et al.* (51).

(*iii*) The mutation links tightly two polymorphic loci that were hitherto far apart in the genome, thereby creating a particular combination of alleles, i.e., a supergene (52), that is unlikely to be destroyed by recombination.

CONCLUSIONS

Our quantitative studies of speciation rates and rates of chromosomal evolution contribute to a better understanding of the mechanism for evolutionary change. The results reported here for vertebrates and elsewhere for seed plants (14) provide a quantitative test for the well-known theory proposed by Wright (1, 2) that adaptive evolution and speciation take place fastest in those species with a marked propensity for population subdivision or deme formation.

The propensity to form small demes is attributable to several factors, one of which may be especially important for understanding how mammals have achieved remarkably high rates of adaptive evolution. This factor is social structuring. If it were not for this social factor, mammals, because of their high dispersal power, might have evolved at the same rate or more slowly than most lower vertebrates.

Propensity to form small demes does not ensure that a species will evolve rapidly. New ecological opportunities must arise as well. Given that a new niche becomes available, however, it seems probable that species composed of small demes will speciate and adapt to the niche faster than those species with large panmictic populations.

Another outcome of our studies concerns the contrast that exists between molecular evolution and evolution at other levels of biological organization. Whereas most mammals have been evolving much faster than most lower vertebrates in karyotype and phenotype and with regard to speciation, there is increasing evidence that the rate at which point mutations have been accumulating in structural genes and unique DNA sequences is not greater in mammals than in lower vertebrates (53). There appear to be two types of evolutionary process, each obeying a different set of rules. It is suggested that population structure and dynamics have little influence on rates of evolutionary substitution in structural genes and unique DNA sequences, though having a profound influence on evolution at other levels.

1. Wright, S. (1931) *Genetics* 16, 97–159.
2. Wright, S. (1940) *Am. Nat.* 74, 232–248.
3. Mayr, E. (1970) *Populations, Species and Evolution* (Harvard Univ. Press, Cambridge, MA).
4. Carson, H. L. (1971) *Stadler Genet. Symp.* 3, 51–70.
5. Eldredge, N. & Gould, S. J. (1972) in *Models in Paleobiology*, ed. Schopf, T., (Freeman, San Francisco, CA), pp. 82–115.
6. Nei, M. (1976) in *Population Genetics and Ecology*, eds. Karlin, S. & Nevo, E. (Academic Press, New York), pp. 723–765.
7. Bush, G. L. (1975) *Annu. Rev. Ecol. Syst.* 6, 339–364.
8. Wilson, A. C., Bush, G. L., Case, S. M. & King, M.-C. (1975) *Proc. Natl. Acad. Sci. USA* 72, 5061–5065.
9. Wright, S. (1941) *Am. Nat.* 75, 513–522.
10. White, M. J. D. (1968) *Science* 159, 1065–1070.
11. White, M. J. D. (1973) *Animal Cytology and Evolution* (Cambridge University Press, England), third edition.
12. Paull, D., Williams, E. E. & Hall, W. P. (1976) *Breviora Mus. Comp. Zool.* 441, 1–31.
13. Stanley, S. M. (1975) *Proc. Natl. Acad. Sci. USA* 72, 646–650.
14. Levin, D. A. & Wilson, A. C. (1976) *Proc. Natl. Acad. Sci. USA* 73, 2086–2090.
15. Wilson, A. C., Sarich, V. M. & Maxson, L. R. (1974) *Proc. Natl. Acad. Sci. USA* 71, 3028–3030.
16. Bickham, J. W. & Baker, R. J. (1976) *Chromosoma* 54, 201–219.
17. Dutrillaux, B. (1976) *Monogr. Anal. Genet.* 3, 1–104.
18. Denton, T. E. (1973) *Fish Chromosome Methodology* (Charles C Thomas, Springfield, IL).
19. Chiarelli, A. B. & Capanna, E., eds. (1973) *Cytotaxonomy and Vertebrate Evolution* (Academic Press, London).
20. Savage, D. E. (1975) "Approaches to primate paleobiology," in *Contr. Primatology*, ed. Szalay, F. S. (Karger, Basel), Vol. 5, pp. 2–27.
21. Gorham, S. W. (1974) *Checklist of World Amphibians up to January 1, 1970* (New Brunswick Museum, St. John, Canada).
22. Anderson, S. & Jones, J. K., Jr. (1967) *Recent Mammals of the World. A Synopsis of Families* (Ronald Press, New York).
23. Kurtén, B. (1972) in *Calibration of Hominoid Evolution*, eds. Bishop, W. W. & Miller, J. A. (Univ. of Toronto Press, Toronto).
24. Lewis, K. R. & John, B. (1963) *Chromosome Marker* (Churchill, London).
25. Arnason, U. (1972) *Hereditas* 70, 113–118.
26. Wiens, J. A. (1976) *Annu. Rev. Ecol. Syst.* 7, 81–120.
27. Struhsaker, T. T. (1969) *Folia Primatol.* 11, 90–118.
28. Kingdon, J. (1971) *East African Mammals* (Academic Press, London and New York), Vol. I.
29. Anderson, P. K. (1970) *Symp. Zool. Soc. London* 26, 299– 325.
30. Wahrman, J., Goitein, R. & Nevo, E. (1969) *Science* 169, 82–84.
31. Pearson, O. P. (1959) in *Peru Mem. Museo Hist. Nat. "Javier Prado"*, 9, 1–56.
32. Patton, J. L. (1973) *Evolution* 26, 574–586.
33. Thaeler, C. S., Jr. (1968) *Chromosoma* 25, 172–183.
34. Reig, O. A. & Kiblisky, P. (1969) *Chromosoma* 28, 211–244.
35. Klingel, H. (1974) in *The Behavior of Ungulates and Its Relation to Management*, eds. Geist, V. & Walter, W. [IUCN Publ. (New Series)], Vol. 24, pp. 124–132.
36. Feist, J. D. & McCullough, D. (1976) *Z. Tierpsychol.* 41, 337–371.
37. Lidicker, W. Z. (1976) *J. Anim. Ecol.*, 45, 677–697.
38. DeFries, J. C. & McClearn, G. E. (1972) *Evol. Biol.* 5, 279–291.
39. Selander, R. K. (1970) *Am. Zool.* 10, 53–66.
40. Brown, L. E. (1969) *Oecologia* 2, 198–222.
41. Rasmussen, D. J. (1970) *Symp. Zool. Soc. London* 26, 335–349.
42. Gropp, A., Winking, A., Zech, L. & Mueller, C. H. (1972) *Chromosoma* 39, 265–288.
43. Capanna, E., Cristadi, M., Perlicone, P. & Rissoni, M. (1975) *Experientia* 31, 294–296.
44. Jotterand, M. (1972) *Rev. Suisse Zool.* 79, 287–359.
45. Bertram, B. C. R. (1975) *Sci. Am.* 232, (May), 54–65.
46. Wurster-Hill, D. H. & Gray, C. W. (1975) *Cytogenet. Cell Genet.* 15, 306–331.
47. Wilson, E. O. (1975) *Sociobiology* (Harvard Univ. Press, Cambridge, MA).
48. Selander, R. K. (1976) in *Molecular Evolution*, ed. Ayala, F. J. (Sinauer Assoc., Sunderland, MA), pp. 21–45.
49. Nei, N., Fuerst, P. A. & Chakraborty, R. (1976) *Nature* 262, 491–493.
50. Grant, V. (1973) *Plant Speciation* (Columbia Univ. Press, New York).
51. Zieg, J., Silverman, M., Hilmen, M. & Simon, M. (1977) *Science* 196, 170–172.
52. Dobzhansky, T. (1970) *Genetics of the Evolutionary Process* (Columbia Univ. Press, New York), third edition.
53. Wilson, A. C., Carlson, S. S. & White, T. J. (1977) *Annu. Rev. Biochem.* 46, 573–639.